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Research Report

Differential modulation of thalamic neurons by optokinetic nuclei in the pigeon

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ABSTRACT

The visual system in the pigeon is composed of the tectofugal, thalamofugal and accessory optic pathways. Though their anatomy and physiology have been extensively studied, the functional interactions between these pathways are largely unknown. The present study shows by using multiple electrophysiological techniques that firing activity in the nucleus opticus principalis thalami (OPT) of the thalamofugal pathway is differentially modulated by the pretectal nucleus lentiformis mesencephali (nLM) and the nucleus of the basal optic root (nBOR) of the accessory optic system, two optokinetic nuclei responsible for generating eye movements to stabilize the image on the retina. Reversible inactivation, electrical stimulation, microiontophoresis and receptive field mapping experiments all consistently indicate that the nBOR-OPT pathway is inhibitory and mediated by GABA as a transmitter and its $GABA_A$ receptors, whereas the nLM-OPT pathway is excitatory and mediated by glutamate as a transmitter and its NMDA receptors. They also differentially modulate the size and/or responsiveness of receptive fields in OPT cells as well. Numerous electrode tip sites were histologically confirmed in the neural structures under study. The results suggest that these optokinetic nuclei may dually modulate the transfer of visual information from the retina to the telencephalon at the thalamic level during eye movements.

1. Introduction

The visual system in birds consists of the tectofugal, thalamofugal and accessory optic pathways, which are homologous to the colliculo-pulvinar-cortical, geniculocortical and accessory optic pathways in mammals, respectively (Karten, 1969; Shimizu and Bowers, 1999). The thalamofugal pathway in birds goes from the retina to the nucleus opticus principalis thalami (OPT) in the thalamus to the telencephalon; this thalamic nucleus is also designated as the nucleus geniculatus lateralis pars dorsalis (Gunturkun and Karten, 1991; Karten et al., 1973) and thought to be homologous to the lateral geniculate nucleus in mammals. Visual cells in the avian OPT are characterized by a large receptive field and selectivity for the direction and speed of object motion (Britto et al., 1975; Jassik-Gerschenfeld et al., 1976; Yang et al., 2005). In addition to afferents from other brain regions, OPT also receives inputs from the pretectal nucleus lentiformis mesencephali (nLM) and the nucleus of the basal optic root (nBOR) of the accessory optic system (Wild, 1989; Wylie et al., 1998), which are homologous to the nucleus of the optic tract and the terminal nuclei of the accessory optic

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tract in mammals, respectively (Fite, 1985; McKenna and Wallman, 1985). Both nLM and nBOR are involved in generating optokinetic nystagmus, an oculomotor reflex for stabilizing the image on the retina by slow-following and saccadic movements of the eyes (Gioanni et al., 1983, 1984; McKenna and Wallman, 1981). Optokinetic neurons are sensitive to the direction, speed and acceleration of visual motion (Cao et al., 2004; Crowder and Wylie, 2002; Frost et al., 1990; Fu et al., 1998; Wolf-Oberhollenzer and Kirschfeld, 1994; Zhang et al., 1999).

Though these visual pathways in birds have been extensively investigated anatomically and physiologically, their functional interactions are still largely unknown. The present study attempted to explore whether and how the accessory optic system would modulate the thalamofugal pathway in general and the optokinetic nuclei, nLM and nBOR, would modulate firing activity of OPT cells in particular, by using single unit recording, reversible inactivation, microiontophoresis, electrical stimulation and receptive field mapping techniques. In some experiments, the electrode tip sites for extracellular recording, drug injection and electrical stimulation were marked for their histological verification.

2. Results

The present study provided several lines of evidence that the nBOR-OPT pathway in pigeons is inhibitory, whereas the nLM-OPT pathway is excitatory, and these pathways differentially modulate visual activity in OPT cells. Its main findings are described in two parts as follows. The first part contained 144 OPT cells that were examined for firing activity modification by pharmacological manipulations of nBOR and nLM; the second part included 36 OPT cells that were examined for receptive field modification by these manipulations. Finally, 44 electrode tip sites for recording, drug application and electrical stimulation were marked with either dye staining or electrolytic lesion to histologically verify their locations in these structures.

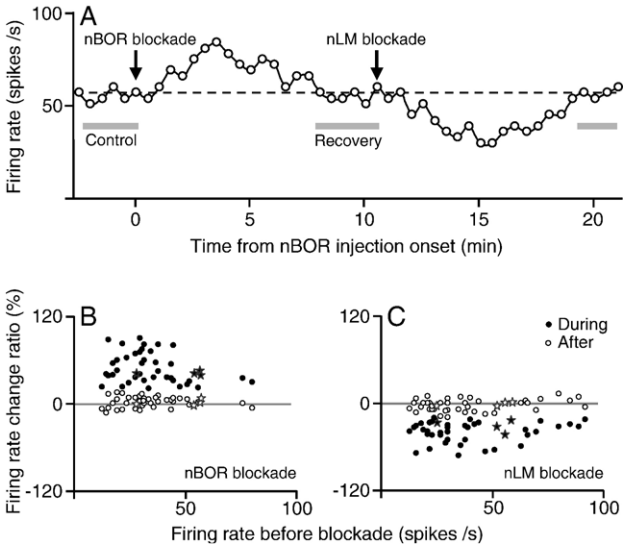

Fig. 2 – Afferents from nBOR and nLM converge onto the same OPT neurons. Visual responses of an OPT cell were increased by nBOR blockade (lidocaine, 90 nl) and then decreased by nLM blockade (GABA, 90 nl) (A). Three repeats are averaged. Statistical analysis of 43 OPT cells in each of two groups shows the change ratio of firing rate (see the Experimental procedures) during (solid symbols) and after (empty ones) blockade of activity in nBOR (B) and nLM (C), indicating that firing rate in these cells were significantly increased by nBOR blockade and decreased by nLM blockade, and the firing activity returned to normal after blockade. Stars represent four OPT cells showing convergence of nBOR and nLM inputs on the same OPT cells examined as in panel A.

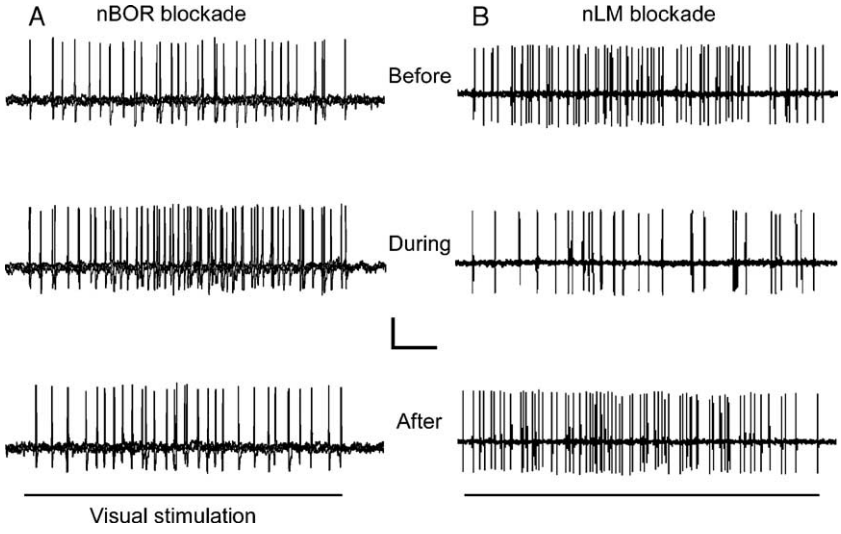

Fig. 1 – Changes in visual responses of two OPT neurons before, during and after blockade of activity in nBOR and nLM. The firing rate of cell A was increased by 48% during nBOR blockade by lidocaine (90 nl), whereas that of cell B decreased by 47% during nLM blockade by GABA (90 nl). Their firing rates were recovered to control values 5–8 min after blockade. Horizontal lines symbolize duration of visual stimulation. Three repeats were superimposed. Scales: 100 ms, 50 μV.

Firing activity modification of 86 out of 144 OPT cells in two groups was examined before, during and after blockade of activity in nBOR and nLM (Fig. 1). During injection of lidocaine in nBOR, the firing rate in a group of 43 OPT cells examined was increased by 50 ± 23%. In contrast, injecting GABA into nLM in an additional group of 43 OPT cells decreased the firing rate by 40 ± 14%. To examine whether afferents from both nBOR and nLM would converge onto the same OPT cells, changes in firing activity in four of these OPT cells were analyzed by sequentially blocking activity in nBOR and nLM. Their firing rate was increased by 48% on average during nBOR blockade and decreased by 34% during nLM blockade. Fig. 2A shows an example of the sequential blockade experiments, confirming a convergence of inhibitory and excitatory inputs from nBOR and nLM on the same OPT cells. Statistical data on the change ratio (see the Materials and methods) in firing rate of these 86 OPT cells before, during and after activity blockade in nBOR and nLM are shown in Figs. 2B and C. Paired *t* tests showed significant change in firing rate during blockade ($P < 0.01$) and no change after blockade ($P > 0.1$) in comparison with the firing rate before blockade as control.

Furthermore, the finding that the nBOR-OPT pathway is inhibitory and nLM-OPT pathway is excitatory was also confirmed by subsequent electrical stimulation and microiontophoresis experiments on 58 of out of 144 OPT cells in two groups. Electrical stimulation of nBOR completely inhibited visual responses in a group of 26 OPT cells for a period of 48.7 ± 9.7 ms with an average latency of 22.2 ± 12.5 ms. This inhibition was removed by bicuculline (20–30 nA) as an antagonist to $GABA_A$ receptors but not by 2-hydroxysaclofen as an antagonist to $GABA_B$ receptors even at a current up to 100 nA and returned to control levels within 3–5 min after bicuculline application (Fig. 3A). On the other hand, electrical stimulation of nLM evoked spiking activity in an additional group of 32 OPT cells with an average latency of 7.2 ± 1.6 ms, suggesting an excitatory contact of nLM cells with OPT cells. Each stimulation of nLM could generally elicit one spike in an OPT cell under control conditions, and the number of spikes evoked by one stimulation was increased to 2–3 during glutamate application (10–20 nA) in OPT cells. However, acetylcholine (10–100 nA) did not work in the nLM-OPT pathway. The spiking activity evoked by nLM stimulation was abolished by CPP (20–60 nA) as an antagonist to NMDA receptors but not by CNQX as an antagonist to AMPA receptors even at a higher dosage (200 nA) (Figs. 3B, C). Meanwhile, we noted that the retino-OPT pathway is also glutamatergic but mediated by AMPA receptors instead because visual responses in OPT cells were abolished by CNQX (20–60 nA) but not by CPP (up to 200 nA) as shown in Fig. 3D.

Receptive field modification by pharmacological manipulations of nBOR and nLM was examined on 36 OPT cells. Computer-aided mapping of receptive fields showed that 61% of these cells possessed a single ERF (E-type) (Figs. 4B, D) and 39% of the cells had an ERF surrounded by an IRF (EI-type) (Figs. 4A, C). We then examined the effect of nBOR blockade on receptive fields in 18 of these 36 OPT cells (11 E-type, 7 EI-type cells) and found that ERF of E-type cells did not change in size (*t* test, $t = 0.92$, $P = 0.61$), whereas ERF of EI-type cells was increased in size by 29 ± 6%. In 18 others (11 E-type, 7 EI-type) examined for the effect of nLM blockade, ERF size of E-type cells did not change (*t* test, $t = 0.90$, $P = 0.39$) and that of EI-type

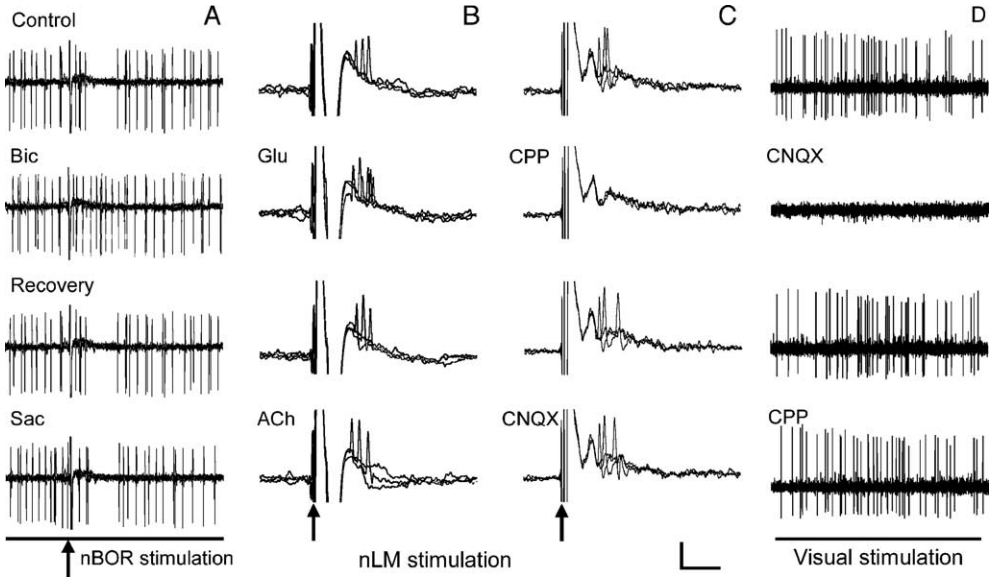

Fig. 3 – Identification of putative transmitters and receptor subtypes in the nBOR- and nLM-OPT pathways. Electrical stimulation of nBOR inhibited visual responses of an OPT cell, and this inhibition was abolished by bicuculline but not by 2-hydroxysaclofen (A). Electrical stimulation of nLM elicited spiking in OPT cells, and this spiking activity was enhanced by glutamate but not by acetylcholine (B). Spiking was eliminated by CPP but not by CNQX (C). Visual responses of an OPT cell to stimulus motion were blocked by CNQX but not by CPP (D). Three repeats are superimposed in panels B and C. Arrows point to electrical stimulation artifacts, horizontal lines mark visual stimulation. Scale bars: 40 ms (A), 5 ms (B, C) and 50 μV (A–C); 100 ms and 25 μV (D).

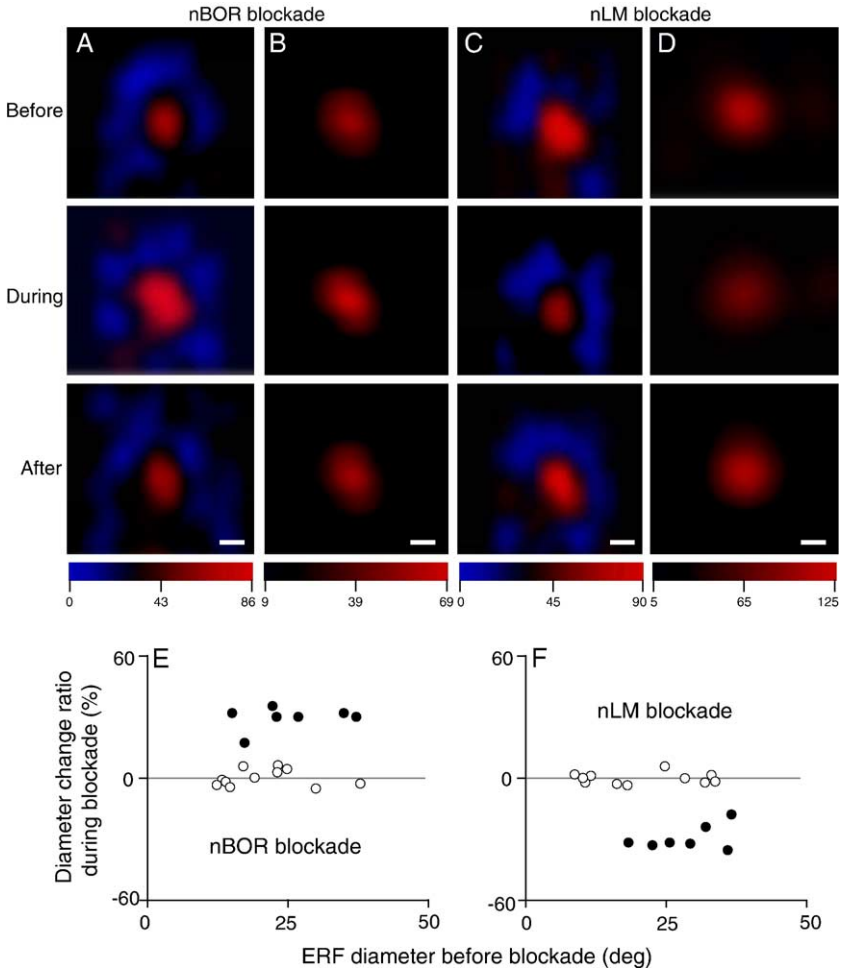

Fig. 4 – Excitatory (red) and inhibitory (blue) receptive fields of four OPT cells (A–D) were modulated by nBOR and nLM. The size of ERF in E-type cells was not changed during blockade of nBOR and nLM (B, D), whereas the size of ERF in EI-type cells was enlarged by nBOR blockade (A) and reduced by nLM blockade (C). Statistical analysis in panels E and F shows dependence of changes in ERF size on the cell type and region being blocked. Solid and empty circles symbolize EI-type and E-type cells, respectively. Color scales represent firing rates in spikes/s and scale bars represent 20, 10, 18 and 13° from panels A to D.

cells was decreased by 28 ± 7% (Figs. 4E, F). Meanwhile, the responsiveness of ERF in both types of OPT cells was enhanced by blockade of nBOR and reduced by that of nLM. Changes in size and responsiveness of IRF in OPT cells during activity blockade in nBOR and nLM were difficult to be measured and therefore not included in the quantitative analysis.

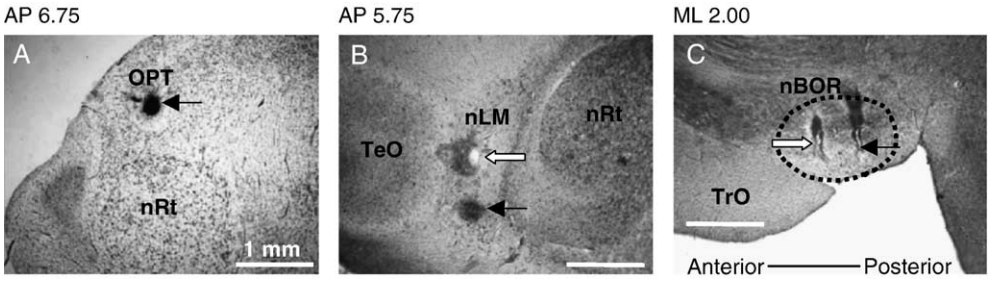

Fig. 5 – Microphotographs of coronal (A, B) and sagittal (C) sections of pigeon's brain mark the locations of electrode sites. The recording site of a thalamic cell was marked with dye within the nucleus opticus principalis thalami (A, OPT, arrow). Both active (solid arrows) and reference (empty arrows) electrodes were marked by electrolytic lesions in the nucleus lentiformis mesencephali (B, nLM) and the nucleus of the basal optic root (C, nBOR). Other abbreviations: nRt, Nucleus rotundus; TeO, Optic tectum; Tro, Optic tract. AP = anterior–posterior levels, ML = mediolateral level of the pigeon brain atlas. Scale bar: 1 mm.

Forty-four electrode sites were histologically localized with either dye or lesion, including 22 recording sites in OPT, 6 drug injection sites and 5 electrical stimulation sites in nBOR, and 6 injection and 5 stimulation sites in nLM. Twenty-two sites marked in OPT were distributed in its subdivisions including 5 in the nucleus dorsolateralis anterior thalami pars magnocellularis (DLAmc), 11 in the nucleus dorsolateralis anterior thalami pars lateralis (DLL) and 6 in the nucleus lateralis anterior thalami (LA). The sites marked in nBOR included 8 in the nBOR proper and 3 in the nBOR pars dorsalis. Among the sites marked in nLM, 7 were in the nucleus lentiformis mesencephali pars medialis and 4 in the nucleus lentiformis mesencephali pars lateralis. In some cases, the active and reference tips of bipolar electrodes were both lesioned, and they were all located within nLM and nBOR (Fig. 5).

3. Discussion

The present study provided four lines of evidence that firing activity in OPT cells of the thalamofugal pathway is differentially modulated by afferents from nBOR and nLM of the accessory optic system. Our experiments using reversible inactivation, electrical stimulation, microiontophoresis and receptive field mapping techniques all consistently indicate in the pigeon that the nBOR-OPT pathway is inhibitory, whereas the nLM-OPT pathway is excitatory.

Previous studies have shown that nBOR can modulate firing activity of rotundal cells in the tectofugal pathway (Diekamp et al., 2001; Wang et al., 2000). The present study indicates for the first time that nBOR also modulates firing activity of OPT cells in the thalamofugal pathway. Several lines of evidence consistently support the notion that the nBOR-OPT pathway (Wylie et al., 1998) is inhibitory. First, blockade of activity in nBOR by lidocaine increased visual responses of OPT cells. Lidocaine as a sodium-channel blocker is a powerful tool for reversible inactivation of firing activity in neural structures (Crowder et al., 2004; Ferrera et al., 1994; Gu et al., 2001; Wang et al., 1995). The fact that drug application sites were marked with dye within nBOR and OPT activity was increased by nBOR blockade strongly implies that lidocaine in nBOR did not influence the optic tract near nBOR. Second, electrical activation of nBOR inhibited visual responses in OPT cells. The placement of stimulating electrodes within nBOR was verified by two facts: all lesioned marks were located within nBOR even though both poles of a stimulating electrode were concurrently lesioned; a decrease in firing rate of OPT cells elicited by nBOR activation is in accordance to an increase in firing activity of OPT cells evoked by nBOR blockade. Third, the inhibition of OPT activity produced by electrical stimulation of nBOR could be removed by bicuculline, characterizing that the nBOR-OPT pathway is GABAergic and mediated by $GABA_A$ receptors. Fourth, blockade of nBOR activity increased both responsiveness and size of ERF in EI-type OPT cells and the responsiveness of ERF in E-type OPT cells as well.

In contrast, the nLM-OPT pathway (Wild, 1989; Wylie et al., 1998) is excitatory because blockade of nLM activity by GABA decreased firing rate of OPT cells. It appears that nLM could keep the excitability or responsiveness of OPT cells. Using GABA instead of lidocaine as a blocker here is due to the fact that GABA specifically blocks nLM cells with GABA receptors but spares the axon fibers passing through nLM from nBOR to OPT (Wylie et al., 1998). This result is consistent with that obtained by electrical stimulation of nLM, which evoked spikes in OPT cells with a short latency and a ratio of one stimulation to one spike. The spiking activity in OPT cells evoked by nLM activation was enhanced by glutamate and depressed by CPP as an antagonist to NMDA receptors, indicating that the nLM-OPT pathway is glutamatergic and mediated by NMDA receptors. In contrast to nBOR blockade, nLM blockade decreased both responsiveness and size of ERF in EI-type OPT cells and the responsiveness of ERF in E-type OPT cells. It appears that differences in changes of ERF size between E- and EI-types of OPT cells may be probably due to the local thalamic circuits that underlie different receptive field organizations but not related to the synaptic nature of the nBOR- and nLM-OPT pathways. The finding that drug application and electrical stimulation sites were all marked within nLM and the effects of nLM manipulations on activity of OPT cells were quite consistent strongly suggests that all these manipulations within nLM did not involve the optic tectum nearby. All these data exclude the possibility that nLM might disinhibit OPT cells although nLM affected the firing activity but not ERF size of E-type OPT cells. Furthermore, the results obtained by various examinations on EI-type OPT cells also support the notion that nLM exerts an excitatory action on OPT cells. It appears that the nLM-thalamic pathway in non-mammals such as pigeons is excitatory, whereas the pretectum in mammals exerts an excitatory action on the relay cells in LGN by disinhibition via GABAergic interneurons (Fischer et al., 1998; Sherman and Guillery, 1996; Wang et al., 2002).

On the other hand, nBOR and nLM are reciprocally connected. The nLM-nBOR pathway is mainly excitatory (Wang et al., 2001), whereas the nBOR-nLM pathway is predominantly inhibitory (Baldo and Britto, 1990; Gu et al., 2001). It is likely that nBOR and nLM may also exert some effects on OPT cells through nLM and nBOR, respectively. Because nBOR and nLM are both involved in generating optokinetic nystagmus for stabilizing the image on the retina by slow-following and saccadic movements of the eyes, the present results suggest that these two optokinetic nuclei may dually modulate visual responses of OPT cells and thus modulate the transfer of visual information from the retina to the telencephalon at the thalamic level during eye movements. Our preliminary results in ongoing experiments show that nBOR and nLM are able to modulate saccadic suppression of OPT cells in an inhibitory and excitatory fashion, respectively.

4. Experimental procedures

Forty-one adult pigeons (*Columba livia*) were used following the guidelines regarding the care and use of animals established by the Society for Neuroscience. Each pigeon was anesthetized with urethane (20%, 1 ml/100 g body weight) and placed in a stereotaxic apparatus. The left rostral tectum and caudal forebrain were exposed, and the dura mater overlying OPT, nLM and nBOR was

excised. The right eye was kept open, and the left was covered. A screen of 130° × 140° was positioned 40 cm away from and tangential to the viewing eye. Because the angle between the horizontal axis of the visual field and the eye center-bill tip line of the stereotaxically fixed pigeon is 72°, while it is 34° under the pigeon's normal conditions (Erichsen et al., 1989), the horizontal axis was thus rotated by 38° (Britto et al., 1990; Fu et al., 1998) to meet the normal conditions.

Visual stimuli were generated by a computer with graphics-card (Ti 4600, MicroStar Co) and back-projected with a projector (PG-M20X, Sharp Co) on the screen. A rectangle (10 × 20°) was moved at 64°/s to evoke visual responses in OPT cells. For mapping the excitatory (ERF) and inhibitory receptive fields (IRF) of OPT cells with the computer, a black square (8°) was moved at 64°/s on the screen randomly along a series of parallel paths covering the whole screen (Fu et al., 1998). The receptive field extent was determined by the equal-rate line of 20% higher (ERF) or lower (IRF) than the average spontaneous rate with software Micrografx Picture Publisher (7a, Micrografx Inc), and its size was measured by averaging the largest and smallest diameters. In non-spontaneous cells, twin-squares (8° each) were used to measure IRF, one of which (control) was moved within ERF and the other (test) moved in the region outside ERF. Both stimuli were moved at the same velocity in the same direction with an increasing distance between. The luminance of black and white in visual stimuli was 0.1 and 6.6 cd/m^2, respectively.

A micropipette (~2-μm tip diameter) filled with 2 M sodium acetate and 2% pontamine skyblue (Gu et al., 2001; Hellon, 1971) was used for extracellular recording and marking electrode tip sites. In some experiments, two-barrel micropipettes were used one barrel of which was filled as above for recording and marking and the other was connected to a pneumatic picopump (PV800, Medical Systems Corp) and contained lidocaine hydrochloride (2%) or GABA (γ-aminobutyric acid, 100 mM, pH 3.3) for blocking nBOR and nLM activity by pressure injections (80–120 nl) (Li et al., 1998). Spatial overlap of the receptive field of an OPT cell with that of an nLM or nBOR cell was a precondition for examining the functional interaction between these cells in different neural structures. For identifying putative transmitters and receptors in the nBOR- and nLM-OPT pathways, a five-barrel micropipette was used one barrel of which was filled as the recording pipette, and the others contained the following compounds (all from Sigma Chemical Co) to be ejected by appropriate currents: sodium-L-glutamate (0.5 M, pH 7.3), CNQX (6-cyano-7-nitroquinoxaline-2, 3-dione, 10 mM, pH 8.3), CPP (3-rs-2-carboxypiperazin-4-yl-propyl-1-phosphonic acid, 10 mM, pH 7.5), acetylcholine chloride (0.5 M, pH 3.5), bicuculline methiodide (10 mM, pH 3.0) or 2-hydroxysaclofen (20 mM, pH 3.0) (Xiao et al., 1999). To activate these pathways, electrical stimulation was delivered to nLM and nBOR with a tungsten bipolar electrode whose poles were glass-coated, ~400 μm apart and ~60 μm tip exposed. Its poles were arranged dorsoventrally and inserted laterally into nLM, whose maximal dimension is ~1500 μm dorsoventrally, to avoid stimulating the optic tectum, or arranged rostrocaudally and inserted vertically into nBOR, which is ~1000 μm rostrocaudally, to avoid stimulating the optic tract. Rectangular pulses of 100–500 μA in intensity and 50–100 μs in duration were applied. The field potential and spikes evoked by electrical stimulation verified a neuronal connection between the recorded cell and the stimulation site. All electrodes were inserted according to the pigeon's brain atlas (Karten et al., 1967).

Action potentials were amplified and fed into an oscilloscope for display and stored in the computer for subsequent data analysis. The interval between trials was at least 10 s to allow the cell to recover from any adaptation. Spikes of OPT cells were collected before, during and after electrical stimulation or pharmacological blockade of nBOR and nLM. Firing rate was obtained by averaging spikes accumulated in 3–10 repeats. The change ratio of firing rate was defined as $(f_1 - f_2)/(f_2 - f_s)$ where f_1 is the firing rate during or after blockade, f_2 is the firing rate before blockade as control, and f_s is the spontaneous rate. Paired t tests showed significant change ($P < 0.01$) or no change ($P > 0.1$) in the firing rate during or after blockade in comparison with the firing rate before blockade.

Recording and drug application sites were marked by dye applied with negative current pulses of 10–20 μA in intensity and 0.5 s in duration at 1 Hz for 10–15 min. Electrical stimulation sites were lesioned by positive currents of 30–40 μA for 10–25 s through the active pole (Wang and Matsumoto, 1990). In some cases, both active and reference poles of bipolar electrodes were lesioned for confirming that electrical stimulation was totally applied within the stimulated structure. Under deep anesthesia, the pigeon was sacrificed, and its brain was removed from the skull, fixed in 4% paraformaldehyde for 6–12 h and soaked in 30% sucrose solution in a refrigerator overnight. Frozen sections were cut at 40 μm and counterstained with cresyl violet. The sections were histologically processed for microscopic observation.

Acknowledgments

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REFERENCES

Baldo, M.V., Britto, L.R., 1990. Accessory optic-pretectal interactions in the pigeon. Braz. J. Med. Biol. Res. 23, 1037–1040.

Britto, L.R., Brunelli, M., Francesconi, W., Magni, F., 1975. Visual response pattern of thalamic neurons in the pigeon. Brain Res. 97, 337–343.

Britto, L.R., Gasparotto, O.C., Hamassaki, D.E., 1990. Visual telencephalon modulates directional selectivity of accessory optic neurons in pigeons. Vis. Neurosci. 4, 3–10.

Cao, P., Gu, Y., Wang, S.R., 2004. Visual neurons in the pigeon brain encode the acceleration of stimulus motion. J. Neurosci. 24, 7690–7698.

Crowder, N.A., Wylie, D.R., 2002. Responses of optokinetic neurons in the pretectum and accessory optic system of the pigeon to large-field plaids. J. Comp. Physiol., A 188, 109–119.

Crowder, N.A., Dickson, C.T., Wylie, D.R., 2004. Telencephalic input to the pretectum of pigeons: an electrophysiological and pharmacological inactivation study. J. Neurophysiol. 91, 274–285.

Diekamp, B., Hellmann, B., Troje, N.F., Wang, S.R., Güntürkün, O., 2001. Electrophysiological and anatomical evidence for a direct projection from the nucleus of the basal optic root to the nucleus rotundus in pigeons. Neurosci. Lett. 305, 103–106.

Erichsen, J.T., Hodos, W., Evinger, C., Bessette, B.B., Phillips, S.J., 1989. Head orientation in pigeon: postral, locomotor and visual determinants. Brain Behav. Evol. 33, 268–278.

Ferrera, V.P., Nealey, T.A., Maunsell, J.H., 1994. Responses in macaque visual area V4 following inactivation of the parvocellular and magnocellular LGN pathways. J. Neurosci. 14, 2080–2088.

Fischer, W.H., Schmidt, M., Hoffmann, K.P., 1998. Saccade-induced activity of dorsal lateral geniculate nucleus X- and Y-cells during pharmacological inactivation of the cat pretectum. Vis. Neurosci. 15, 197–210.

Fite, K.V., 1985. Pretectal and accessory-optic visual nuclei of fish, amphibia and reptiles: theme and variations. Brain Behav. Evol. 26, 71–90.

Frost, B.J., Wylie, D.R., Wang, Y.C., 1990. The processing of object and self-motion in the tectofugal and accessory optic pathways of birds. Vision Res. 30, 1677–1688.

Fu, Y.X., Gao, H.F., Guo, M.W., Wang, S.R., 1998. Receptive field properties of visual neurons in the avian nucleus lentiformis mesencephali. Exp. Brain Res. 118, 279–285.

Gioanni, H., Rey, J., Villalobos, J., Richard, D., Dalbera, A., 1983. Optokinetic nystagmus in the pigeon (*Columba livia*): II. Role of the pretectal nucleus of the accessory optic system (AOS). Exp. Brain Res. 50, 237–247.

Gioanni, H., Rey, J., Villalobos, J., Dalbera, A., 1984. Single unit activity in the nucleus of the basal optic root (nBOR) during optokinetic, vestibular and visuo-vestibular stimulations in the alert pigeon (*Columbia livia*). Exp. Brain Res. 57, 49–60.

Gu, Y., Wang, Y., Wang, S.R., 2001. Directional modulation of visual responses of pretectal neurons by accessory optic neurons in pigeons. Neuroscience 104, 153–159.

Gunturkun, O., Karten, H.J., 1991. An immunocytochemical analysis of the lateral geniculate complex in the pigeon (*Columba livia*). J. Comp. Neurol. 314, 721–749.

Hellon, R.F., 1971. The marking of electrode tip positions in nervous tissue. J. Physiol. (London) 214, 12.

Jassik-Gerschenfeld, D., Teulon, J., Ropert, N., 1976. Visual receptive field types in the nucleus dorsolateralis anterior of the pigeon's thalamus. Brain Res. 108, 295–306.

Karten, H.J., 1969. The organization of the avian telencephalon some speculations on the phylogeny of the amniote telencephalon. Ann. N. Y. Acad. Sci. 167, 164–180.

Karten, H.J., Hodos, W., 1967. A Stereotaxic Atlas of the Brain of the Pigeon (*Columba livia*). Johns Hopkins UP, Baltimore.

Karten, H.J., Hodos, W., Nauta, W.J., Revzin, A.M., 1973. Neural connections of the "visual wulst" of the avian telencephalon. Experimental studies in the pigeon (*Columba livia*) and owl (*Speotyto cunicularia*). J. Comp. Neurol. 150, 253–278.

Li, J.L., Xiao, Q., Fu, Y.X., Wang, S.R., 1998. Centrifugal innervation modulates visual activity of tectal cells in pigeons. Vis. Neurosci. 15, 411–415.

McKenna, O.C., Wallman, J., 1981. Identification of avian brain regions responsive to retinal slip using 2-deoxyglucose. Brain Res. 210, 455–460.

McKenna, O.C., Wallman, J., 1985. Accessory optic system and pretectum of birds: comparisons with those of other vertebrates. Brain Behav. Evol. 26, 91–116.

Sherman, S.M., Guillery, R.W., 1996. Functional organization of thalamocortical relays. J. Neurophysiol. 76, 1367–1395.

Shimizu, T., Bowers, A.N., 1999. Visual circuits of the avian telencephalon: evolutionary implications. Behav. Brain Res. 98, 183–191.

Wang, S.R., Matsumoto, N., 1990. Postsynaptic potentials and morphology of tectal cells responding to electrical stimulation of the bullfrog nucleus isthmi. Vis. Neurosci. 5, 479–488.

Wang, S.R., Wang, Y.C., Frost, B.J., 1995. Magnocellular and parvocellular divisions of pigeon nucleus isthmi differentially modulate visual responses in the tectum. Exp. Brain Res. 104, 376–384.

Wang, Y., Gu, Y., Wang, S.R., 2000. Modulatory effects of the nucleus of the basal optic root on rotundal neurons in pigeons. Brain Behav. Evol. 56, 287–292.

Wang, Y., Gu, Y., Wang, S.R., 2001. Directional responses of basal optic neurons are modulated by the nucleus lentiformis mesencephali in pigeons. Neurosci. Lett. 311, 33–36.

Wang, S., Eisenback, M., Datskovskaia, A., Boyce, M., Bickford, M.E., 2002. GABAergic pretectal terminals contact GABAergic interneurons in the cat dorsal lateral geniculate nucleus. Neurosci. Lett. 323, 141–145.

Wild, J.M., 1989. Pretectal and tectal projections to the homologue of the dorsal lateral geniculate nucleus in the pigeon: an anterograde and retrograde tracing study with cholera toxin conjugated to horseradish peroxidase. Brain Res. 479, 130–137.

Wolf-Oberhollenzer, F., Kirschfeld, K., 1994. Motion sensitivity in the nucleus of the basal optic root of the pigeon. J. Neurophysiol. 71, 1559–1573.

Wylie, D.R., Glover, R.G., Lau, K.L., 1998. Projections from the accessory optic system and pretectum to the dorsolateral thalamus in the pigeon (*Columbia livia*): a study using both anterograde and retrograde tracers. J. Comp. Neurol. 391, 456–469.

Xiao, J., Wang, Y., Wang, S.R., 1999. Effects of glutamatergic, cholinergic and GABAergic antagonists on tectal cells in toads. Neuroscience 90, 1061–1067.

Yang, J., Zhang, C., Wang, S.R., 2005. A comparison of visual properties between tectal and thalamic neurons with overlapping receptive fields in the pigeon. Brain Behav. Evol. 65, 33–39.

Zhang, T., Fu, Y.X., Hu, J., Wang, S.R., 1999. Receptive field characteristics of neurons in the nucleus of the basal optic root in pigeons. Neuroscience 91, 33–40.